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Participate in the Pathophysiology of Neurological Diseases

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Abstract

Extracellular vesicles have emerged as a novel communication paradigm of the complement system in neurological diseases. Complement is an essential part of the innate immune system, playing a crucial role in host defense, tissue homeostasis, and immune regulation. However, dysregulation of the complement system has been implicated in the pathogenesis of various neurological disorders, including Alzheimer's disease, multiple sclerosis, and stroke. In recent years, it has become evident that complement components are present in EVs, which are small membranous vesicles secreted by cells into the extracellular space. These EV-associated complement proteins and microRNAs can modulate immune responses, contribute to neuroinflammation, and participate in the pathophysiology of neurological diseases. This article explores the role of EVs as a means of complement communication in neurological diseases and highlights their potential as diagnostic biomarkers and therapeutic targets.

Keywords: Neurological diseases • Pathogenesis • Alzheimer's disease • Demyelinating disorder

Introduction

Extracellular vesicles are a heterogeneous group of membrane-bound vesicles released by cells, encompassing exosomes, microvesicles and apoptotic bodies. Initially regarded as a means of cellular waste disposal, EVs are now recognized as important mediators of cell-to-cell communication. The complement system, a vital component of the immune system, consists of a complex network of proteins that play a crucial role in recognizing and eliminating pathogens and damaged cells. However, excessive or dysregulated complement activation has been implicated in various neurological disorders. Recent research has revealed that complement proteins can be packaged within EVs and released into the extracellular milieu. These EV-associated complement proteins and microRNAs have emerged as key players in intercellular communication in neurological diseases. Understanding the role of EVs in complement communication provides new insights into disease mechanisms and opens up possibilities for targeted therapies and diagnostic approaches. Complement activation has been observed in the brains of individuals with AD, contributing to neuroinflammation and synaptic dysfunction. In AD, amyloid-βplaques can activate the complement system, leading to the formation of membrane attack complexes and subsequent neurodegeneration. EVs released by activated microglia or neurons in AD may carry complement proteins, including C1q, C3 and C9, and propagate complement activation, exacerbating the neuroinflammatory response [1].

Literature Review

MS is an autoimmune demyelinating disorder characterized by immune-mediated damage to the central nervous system. Dysregulation of the complement system contributes to the inflammatory demyelination observed in MS lesions. EVs released by immune cells, including microglia and astrocytes, can carry complement components and influence immune responses within the CNS. These EVs may also transport miRNAs that regulate complement expression and modulate the inflammatory cascade. In ischemic stroke, complement activation occurs in the ischemic brain tissue and exacerbates tissue damage through the formation of MAC and the release of pro-inflammatory cytokines. EVs released by damaged neurons and immune cells may contribute to complement activation and propagation of the inflammatory response following stroke. Furthermore, complement-associated EVs could serve as potential biomarkers for stroke severity and prognosis [2].

Discussion

EVs can carry complement proteins, including and factor H, as well as complement receptors, such as CR1 and CR3. These complement-associated EVs can facilitate complement activation and opsonization of target cells, contributing to the clearance of pathogens and cellular debris. However, dysregulated complement activation within EVs may lead to excessive inflammation and tissue damage in neurological diseases. Complement proteins present on the surface of EVs can engage with complement receptors on recipient cells, leading to EV internalization. This complement-mediated uptake may enhance the delivery of cargo, such as miRNAs or other bioactive molecules, to target cells. Additionally, complement-coated EVs may facilitate the uptake of antigens by antigen-presenting cells, further influencing immune responses. EV-associated complement proteins and miRNAs can influence the polarization and activation of immune cells. For instance, complement-coated EVs can skew microglia towards a pro-inflammatory phenotype, exacerbating neuroinflammation in neurological disorders. On the other hand, miRNAs carried by EVs may regulate the expression of complement genes and impact complement-mediated immune responses [3].

The presence of complement components and miRNAs within EVs presents an exciting opportunity for developing non-invasive diagnostic biomarkers for neurological diseases. EVs can be isolated from various biofluids, including cerebrospinal fluid, blood and urine, allowing for the assessment of disease-specific EV cargo. Complement-associated EVs may serve as biomarkers of complement dysregulation and neuroinflammation, aiding in early disease detection and monitoring disease progression. Targeting EV-mediated complement communication holds promise for therapeutic interventions in neurological diseases. Strategies to modulate complement activation within EVs or alter EV cargo delivery could be explored. For instance, engineering EVs to carry specific miRNAs or therapeutic molecules that regulate complement expression may offer a novel approach to mitigate neuroinflammation. Additionally, EVs may serve as delivery vehicles for complement inhibitors or

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neuroprotective agents, aiming to modulate the inflammatory response and promote tissue repair.

Extracellular vesicles have emerged as a novel communication paradigm of the complement system in neurological diseases. Complement proteins and miRNAs carried by EVs can modulate immune responses, contribute to neuroinflammation, and participate in the pathophysiology of neurological disorders, including Alzheimer's disease, multiple sclerosis, and stroke. Understanding the role of EVs in complement communication provides new insights into disease mechanisms and offers potential avenues for diagnostic biomarkers and therapeutic interventions. Continued research in this area will help unravel the complex interplay between EVs and the complement system, paving the way for innovative approaches to combat neurological diseases. Neurological diseases encompass a wide range of disorders that affect the central and peripheral nervous systems. The complement system, a critical component of the innate immune response, has emerged as a key player in neuroinflammation and neurodegenerative processes. Traditionally, complement activation and signaling were thought to occur primarily through direct cell-to-cell interactions. However, recent research has highlighted the role of extracellular vesicles as a novel mode of complement communication in neurological diseases. EVs, including exosomes and microvesicles, are small membrane-bound particles released by various cell types that mediate intercellular communication by transferring bioactive molecules, including complement components. This article explores the emerging role of EVs as a vehicle for complement signaling in neurological diseases, shedding light on their implications for disease pathogenesis and potential therapeutic interventions [4].

EVs can activate the complement system by serving as a platform for complement component assembly and activation. For example, EV-associated C1q can bind to target surfaces, leading to the formation of the C3 convertase and subsequent complement cascade activation. This process can occur independently of cell-to-cell contact, facilitating complement activation in a localized manner. EVs can transfer complement components from one cell to another, thereby modulating complement signaling in recipient cells. For instance, microglia-derived EVs can deliver complement proteins to astrocytes or neurons, influencing their complement status and contributing to neuroinflammation or neurodegenerative processes. In addition to complement activation, EVs can also regulate complement activity. EV-associated complement regulators, such as CD55 and CD59, can confer protection against complement-mediated damage by inhibiting complement activation on EV surfaces. Conversely, EVs can also promote complement activation by providing a platform for the assembly of complement components and amplification of the cascade [5].

EV-mediated complement communication has been implicated in neuroinflammatory disorders, such as multiple sclerosis and Alzheimer's disease. In MS, EVs derived from activated immune cells can transport complement components across the blood-brain barrier, contributing to local inflammation and demyelination. In AD, EVs carrying complement proteins, particularly C1q, can facilitate amyloid-βdeposition and neuroinflammation, exacerbating disease pathology. EV-associated complement components have been identified in several neurodegenerative diseases, including Parkinson's disease and Amyotrophic Lateral Sclerosis (ALS). EV-mediated complement activation and transfer of complement components can promote neuroinflammation, protein aggregation, and neuronal damage. These processes contribute to the progressive neurodegeneration observed in these disorders. EV-mediated complement signaling has also been implicated in acute neurological insults, such as stroke and TBI. EVs released from damaged cells can activate the complement cascade, exacerbating neuroinflammation and secondary brain injury. Moreover, EV-mediated transfer of complement components can impact the complement status of recipient cells, further influencing the inflammatory response [6].

Conclusion

The emerging understanding of EV-mediated complements communication in neurological diseases opens up new avenues for therapeutic interventions. Targeting EV-mediated complement activation or modulating EV cargo may offer potential strategies to attenuate neuroinflammation, prevent neuronal damage and slow disease progression. Furthermore, EVs themselves can be explored as therapeutic vehicles for delivering complement regulators or modulating complement activity in a targeted manner. Extracellular vesicles have emerged as a novel mode of complement communication in neurological diseases, providing a new perspective on how complement activation and signaling occur. The transfer of complement components through EVs and the ability of EVs to activate or regulate complement signaling have significant implications for neuroinflammation, neurodegenerative processes and acute neurological insults. Understanding the role of EV-mediated complement communication in neurological diseases may lead to the development of innovative therapeutic strategies that target this communication paradigm, ultimately providing new avenues for intervention in these complex and devastating conditions.

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Conflict of Interest

None.

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